IN THE CLAIMS

1. (Presently Amended): A non-human homozygous transgenic animal having a mutated rchd534 gene, wherein the mutated rchd534 gene is a rchd534-LacZ gene which lacks the MH2 domain encoding region, wherein the wild-type rchd534 gene has been replaced with the mutated rchd534 gene ~~a rchd534-LacZ gene which lacks the MH2 domain encoding region~~, and wherein said animal displays a cardiovascular disease symptom.

2. (Previously Presented): The transgenic animal of claim 1, wherein said cardiovascular disease symptom is hyperplasia, thickening of at least one cardiac valve, cardiac outflow tract development defects, cardiovascular calcification, epicardial vascular malformations, endocardial vascular malformation, or defects in the regulation of vascular tone.

3. (Previously Presented): The transgenic animal of claim 1, wherein said cardiovascular disease symptom is cardiovascular calcification.

4. (Previously Presented): The transgenic animal of claim 1, wherein said cardiovascular disease symptom is aortic or valvular calcification.

5. (Presently Canceled)

6. (Previously Presented): A cell having a mutated rchd534 gene isolated from the transgenic animal of claim 1, wherein said cell is isolated from tissue displaying a cardiovascular disease symptom.

7. (Previously Presented): The cell of claim 6, wherein said symptom is hyperplasia, thickening of at least one cardiac valve, cardiac outflow tract development defects, cardiovascular calcification, epicardial vascular malformation, endocardial vascular malformation, or defects in the regulation of vascular tone.

8. (Previously Presented): The cell of claim 6, wherein said symptom is cardiovascular calcification.

9. (Previously Presented): A cell line established from the cell of claim 6, wherein said cell is isolated from a tissue which exhibits at least one of the following cardiovascular developmental phenotypes: hyperplasia, thickening of at least one cardiac valve, cardiac outflow tract development defects, aortic ossification, epicardial vascular malformation, endocardial vascular malformation, or defects in the regulation of vascular tone.

10-13. (Presently Canceled)

14-25. (Previously canceled)

26. (Presently Amended): A method for identifying a substance for treating or preventing cardiovascular disease, comprising administering said substance to a non-human homozygous transgenic animal having a mutated rchd534 gene, wherein the mutated rchd534 gene is a rchd534-LacZ gene which lacks the MH2 domain encoding region, wherein the wild-type rchd534 gene has been replaced with the mutated rchd534 gene ~~a rchd534-LacZ gene which lacks the MH2 domain encoding region~~, wherein said animal displays a cardiovascular disease symptom, and wherein amelioration of said cardiovascular disease symptom indicates a substance effective in the treatment or prevention of cardiovascular disease.

27. (Previously Presented): The method of claim 26, wherein said cardiovascular disease symptom is hyperplasia, thickening of at least one cardiac valve, cardiac outflow tract development defects, cardiovascular calcification, epicardial vascular malformation, endocardial vascular malformation, or defects in the regulation of vascular tone.

28. (Previously Presented): The method of claim 26, wherein said cardiovascular disease symptom is cardiovascular calcification.

29-33. (Previously Canceled)